Clinical Case Report

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Visual loss due to optic nerve infarction and central retinal artery occlusion after spine surgery in the prone position

A case report

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Abstract

Rationale: Visual loss after spine surgery in the prone position is a serious complication. Several cases of central retinal artery occlusion with ophthalmoplegia after spine surgery have been reported in patients with ophthalmic arteries fed by the internal carotid artery (ICA) in a normal manner.

Patient concerns: A 74-year-old man developed visual loss after undergoing a spinal decompression and fusion operation in the prone position that lasted approximately 5 hours.

Diagnoses: We detected an extremely rare case of visual loss due to optic nerve infarction and central retinal artery occlusion through fundoscopic examination, fluorescein angiogram, brain magnetic resonance imaging, and magnetic resonance angiography. The patient's visual loss may have been caused by compromised retrograde collateral circulation of the ophthalmic artery from branches of the external carotid artery in the presence of proximal ICA occlusion after a spinal operation in the prone position.

Interventions: To recover movement of the left extraocular muscles, the patient received intravenous injections of methylprednisolone for 3 days and then oral prednisolone for 6 days.

Outcomes: Twenty days after the treatment, the motion of the left extraocular muscles was significantly improved. However, recovery from the left visual loss did not occur until 4 months after the operation.

Lessons: In high-risk patients with retrograde collateral circulation of the ophthalmic artery from the external carotid artery due to proximal ICA occlusion, various measures, including the use of a head fixator to provide a position completely free of direct compression of the head and face, should be considered to decrease the risk of postoperative visual loss.

Abbreviations: ECA = external carotid artery, ICA = internal carotid artery, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging.

Keywords: central retinal artery occlusion, external carotid artery, internal carotid artery occlusion, optic nerve infarction, prone position, visual loss

1. Introduction

Reports have indicated that perioperative visual loss after spine surgery is attributable to various causes, including hypotension, embolism, hemorrhagic shock, direct trauma, and prolonged compression of the eyes, and the incidence of this complication after spinal fusion is 0.03%.[1] Patients with carotid stenosis or occlusion have a higher risk of visual loss than those without stenosis. Several cases of central retinal artery occlusion with ophthalmoplegia after spine surgery in patients with ophthalmic arteries fed by the internal carotid artery (ICA) in a normal manner have been reported.[2–4] However, to our knowledge, this is the first report of visual loss due to optic nerve infarction and central retinal artery occlusion caused by compromised retrograde collateral circulation of the ophthalmic artery from the external carotid artery (ECA) in a patient with proximal ICA occlusion after spine surgery in the prone position. We also propose a mechanism presumed to be involved in visual loss due to optic nerve infarction and central retinal artery occlusion and suggest helpful measures to reduce visual loss associated with spine surgery.

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2. Case report

A 74-year-old man (body mass index 24.7) was scheduled to undergo L3–4–5-S1 posterolateral fusion with L5-S1 interbody fusion for lower back and radiating leg pain under general anesthesia. He had been diagnosed with hypertension 10 years earlier and a cerebral infarction 5 years earlier. He was taking drugs, including a calcium channel blocker, an angiotensin II receptor antagonist, and clopidogrel, to treat these conditions. In preoperative carotid ultrasonography, the patient exhibited calcified plaques at both carotid bulbs, and his left carotid artery was totally occluded. The results of other laboratory examinations were within normal ranges. After anesthesia was induced via the intravenous administration of sodium thiopental (250 mg), the patient was manually ventilated with 100% oxygen and received intravenously administered vecuronium (10 mg) for muscle relaxation. Orotracheal intubation was performed using a reinforced endothelial tube (size 7.0), and anesthesia was then maintained using oxygen (2 L/min), nitrous oxide (2 L/min), and sevoflurane (1.5–2.5 volume%). The total duration of anesthesia and surgery was 375 minutes. During the intraoperative period, the total fluid input was 1500 mL, and the total urine output was 150 mL. The estimated blood loss during the surgery was 800 mL. Two units of packed red blood cells were transfused during the operation. The patient's blood pressure and heart rate during the operation ranged from 110/52 to 158/75 mm Hg and from 52 to 78 beats per minute, respectively, with the exception of a blood pressure of 95/43 mm Hg that persisted for only 3 minutes. The procedure was uneventful. After recovery from general anesthesia, the patient could not open his left eye because of swelling, and he was transferred to a general ward. At 12 hours after the end of the operation, his left eyelid continued to look edematous and showed ptosis, and visual loss in his left eye was detected. There was no remarkable left eye pain. The patient underwent further ophthalmic and neurologic examinations. An ophthalmic examination indicated that for the left eye, the patient had no perception of light and limited extraocular movement in all directions of gaze, suggesting total ophthalmoplegia. A fundoscopic examination (Fig. 1A) showed a cherry-red spot and an edematous retina. A fluorescein angiogram of the left eye (Fig. 1B) showed delayed arterial filling. Brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) were performed to exclude perioperative cerebral infarction. The patient's brain MRI showed multiple focal chronic infarctions, but no acute lesions. However, the extraocular muscles and periorbital tissue of the left eye showed diffuse swelling and increased signal intensity on T2-weighted images (Fig. 2A), and the optic nerve was bright on diffusion-weighted images (Fig. 2B), suggesting acute optic nerve infarction. There was segmental occlusion of the left ICA from the cervical segment to the petrous segment, and the patient's left ophthalmic artery was engorged and connected to the distal segment of the proximally occluded left ICA on MRA (Fig. 2C). The feeding of the patient's ophthalmic arterial circulation was estimated to be retrograde, supplied by the branches of the ECA. To recover movement of left extraocular muscles, the patient received intravenous injections of 1 g methylprednisolone for 3 days, with this treatment subsequently switched to oral prednisolone. Oral prednisolone (60 mg) was administered for 3 days and was then tapered to

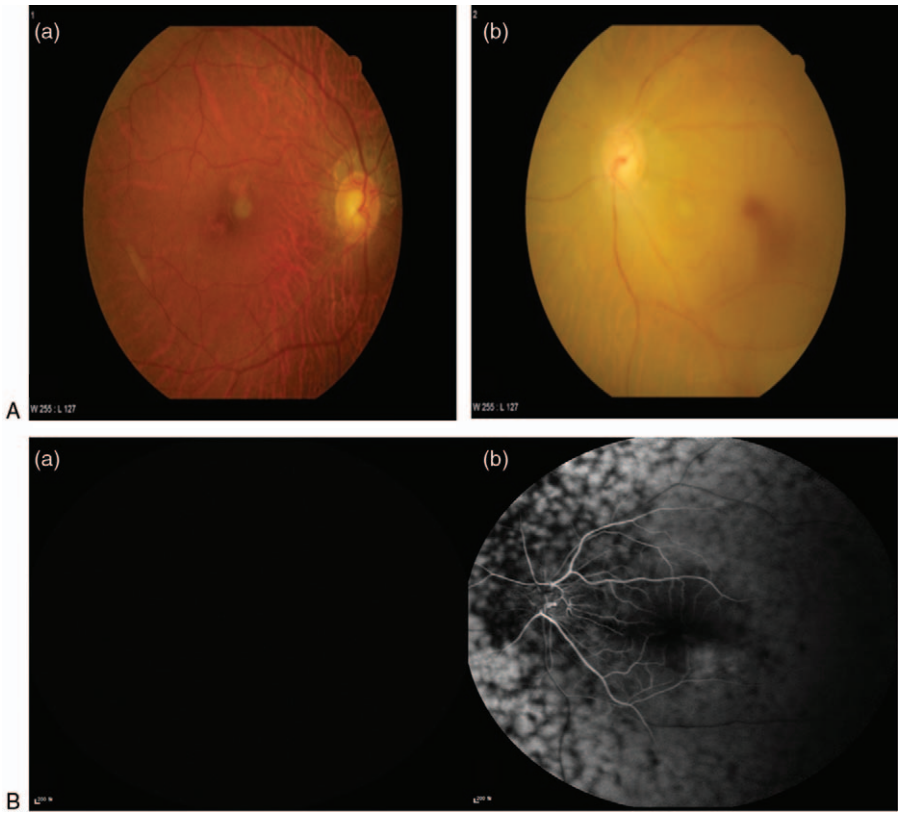

Figure 1. (A) Fundus photographs from the patient's initial visit. (a) The right eye exhibits a normal fundus. (b) The left eye exhibits a pale optic disc with an edematous retina and a cherry-red spot on the fovea. (B) Fluorescein angiograms of the left eye from the patient's initial visit. (a) At 10 seconds after injection, filling of the retina and choroidal vessels is not observed. (b) At 27 seconds after injection, filling of the retinal vein and choroidal vessels remains incomplete.

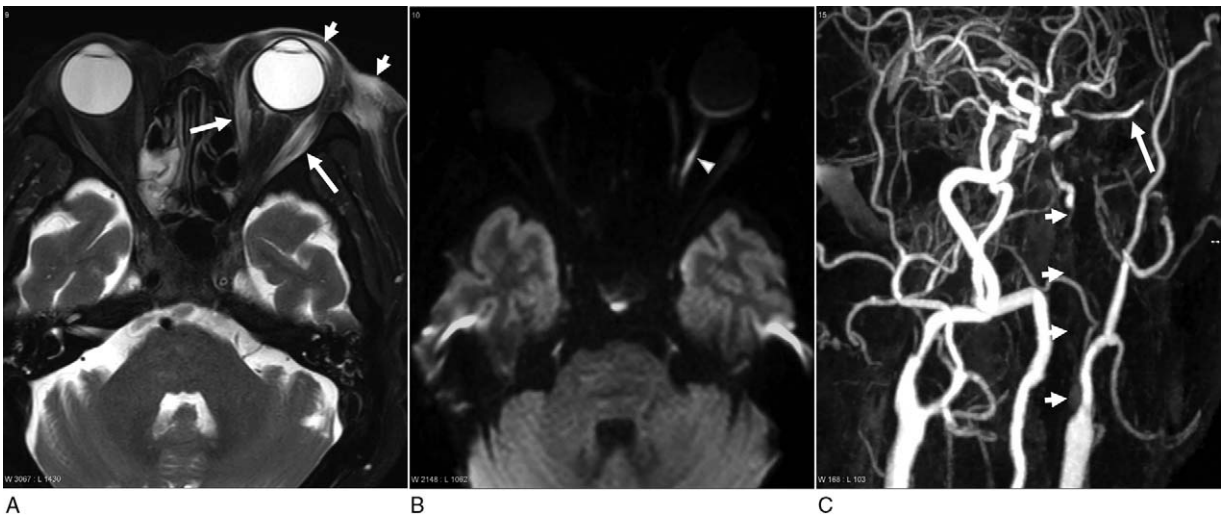

Figure 2. (A) A T2-weighted image shows diffuse swelling and increased signal intensity in the left periorbital soft tissue (short arrows) and rectus muscles (long arrows). (B) On a diffusion-weighted image, the left optic nerve exhibits marked hyperintensity (arrowhead), suggesting infarction. (C) An oblique projection of contrast-enhanced magnetic resonance angiography reveals a long segmental occlusion in the left internal carotid artery from the cervical segment to the petrous segment (short arrows). The left ophthalmic artery (long arrow) is enlarged and might be filled via a collateral pathway from the external carotid artery.

30 mg for 3 additional days. Twenty days after the treatment, the swelling of the patient's left eyelid had markedly decreased, and motion of the left extraocular muscles was significantly improved. However, recovery from left visual loss due to optic nerve infarction and central retinal artery occlusion had not occurred by 4 months after the operation.

3. Discussion

Loss of vision after spine surgery is a serious complication that reduces quality of life. Causes of postoperative visual loss are classified into 4 categories: ischemic optic neuropathy, central retinal artery occlusion, cortical blindness, and posterior reversible encephalopathy syndrome.[1] The most common cause of perioperative visual loss after a spinal fusion operation is ischemic optic neuropathy.[5] According to a report by the Postoperative Visual Loss Study Group of the American Society of Anesthesiologists, independent risk factors related to ischemic optic neuropathy after spinal fusion operations include male sex, obesity, extensive blood loss, prolonged anesthesia, administration of a lower percentage of colloids, and the use of the Wilson frame.[5]

Central retinal artery occlusion reduces blood supply to the retina.[1] Reports have indicated that such occlusion is associated with head position and external compression of the eye, which is accompanied by ptosis and ophthalmoplegia.[1] Human orbits are supplied by the ophthalmic artery, which is a major branch of the ICA.[6] In certain patients with severe ICA stenosis or occlusion, ophthalmic arterial flow can be reversed from antegrade to retrograde; under these circumstances, the ophthalmic artery is supplied by various branches of the ECA, including the facial, maxillary, and superficial temporal arteries.[6] In cases involving proximal ICA occlusion, branches of the ECA provide important collateral blood supply to the ipsilateral ophthalmic artery.[7] Our patient had left proximal ICA occlusion, which was located proximal to the petrous ICA before the branching of the ophthalmic artery normally originated from the ICA (Fig. 2C); thus, based on prior reports, retrograde collateral circulation into the ophthalmic artery via the left ECA was suspected (Fig. 2C).[6,7] In addition, a report indicated that carotid artery stenting in a patient with severe ICA stenosis shifts the blood flow pattern of the ophthalmic artery from retrograde to antegrade, suggesting that retrograde collateral circulation is developed.[8] In our case, a soft, padded, rectangular headrest filled with gel that included space (diameter 12 cm) to rest the forehead and chin was used to provide direct compression-free positioning for the eyes; therefore, direct compression of the eye was relatively unlikely to be involved in central retinal artery occlusion. Thus, compromised collateral circulation from the left ECA, which feeds the ophthalmic artery, was highly likely to contribute to central retinal artery occlusion. Possible causes that may have contributed to compromised retrograde collateral circulation of the left ECA feeding the ophthalmic artery include direct compression of the facial, maxillary, and superficial temporal arteries, which are located in the face, while the patient was in the prone position; increased venous pressure and decreased ophthalmic blood flow due to prolonged time in the prone position; and intraoperative hypotension. Because our patient showed stable intraoperative blood pressures ranging from 110/52 to 158/75 mm Hg, intraoperative hypotension is relatively unlikely to have contributed to his postoperative visual loss. Although we did not confirm the detailed arterial branching of the ECA involved in the retrograde feeding of the ophthalmic artery in our patient, we hypothesized that the branch most likely to be involved is the facial artery. Mechanisms associated with retinal and optic nerve damage produced by orbital infarction syndrome include perfusion failure (common carotid artery occlusion), systemic vasculitis, and orbital cellulitis with vasculitis.[9] Thus, the optic nerve infarction (Fig. 2B) observed in our patient was due to decreased retrograde collateral circulation of the ECA into the ophthalmic artery.[1,9] In addition, increased venous pressure while the patient was in the prone position and venous congestion due to compression may have contributed to central retinal artery occlusion and optic nerve infarction due to decreased retrograde ophthalmic blood flow. In funduscopic examination of the patient, the optic disc was pale, and a cherry-red spot at the fovea was observed, suggesting central retinal artery occlusion (Fig. 1A). The delayed

arterial filling time observed in this patient provided additional evidence of central retinal artery occlusion (Fig. 1B). For high-risk patients with proximal ICA occlusion who must undergo spine surgery in the prone position, intraoperative measures to reduce the possibility of postoperative visual loss should be considered. For this patient with retrograde collateral blood supply to the ophthalmic artery via the ECA, we believe that a more reasonable approach would have been to use a head fixator, such as a Mayfield clamp, to provide completely direct compression-free positioning of the face and eyes during prone position. When abnormal ptosis and ophthalmoplegia are observed after a patient awakens, an ophthalmologist should be consulted as soon as possible. The ophthalmoplegia observed in our patient may have been attributable to compression of the ECA, which helps supply blood to extraocular muscles, and venous congestion induced by the head-down position and facial compression. Postoperative release of the ischemic compression of extraocular muscles may lead to edema and ophthalmoplegia because ischemia-induced vascular channel dilation appears to cause the transudation of fluid into interstitial tissues.[10] As observed in this patient, recovery from ophthalmoplegia is possible because muscle cells are relatively tolerant of ischemic conditions. However, visual loss due to optic nerve infarction and central retinal artery occlusion is typically irreversible, as was the case for our patient.

In conclusion, for high-risk patients with retrograde collateral circulation of the ECA feeding the ophthalmic artery due to proximal ICA occlusion who will undergo spine surgery in the prone position, several measures, including using a head fixator to provide completely direct compression-free positioning of the face and eyes, a less steep head-down position, and preventing intraoperative hypotension, should be considered to reduce the risk of postoperative visual loss due to central retinal artery occlusion and optic nerve infarction.

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